(FILE 'HOME' ENTERED AT 17:33:16 ON 04 AUG 1998)

INDEX 'AGRICOLA, AIDSLINE, ANABSTR, AQUASCI, BIOBUSINESS, BIOSIS, BIOTECHABS, BIOTECHDS, CABA, CANCERLIT, CAPLUS, CEABA, CEN, CIN, CJACS, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGLAUNCH, DRUGNL, DRUGU, EMBAL, EMBASE, FSTA, GENBANK, ...'
ENTERED AT 17:33:28 ON 04 AUG 1998

SEA LIPASE(10W)UREASE

4	FILE ANABSTR
3	FILE AQUASCI
2	FILE BIOBUSINESS
34	FILE BIOSIS
12	FILE BIOTECHABS
12	FILE BIOTECHDS
6	FILE CABA
31	FILE CAPLUS
1	FILE CEABA
1	FILE DDFU
1	FILE DRUGU
6	FILE EMBASE
6	FILE FSTA
8	FILE IFIPAT
6	FILE LIFESCI
5	FILE MEDLINE
5	FILE SCISEARCH
6	FILE TOXLINE
2	FILE TOXLIT
48	FILE USPATFULL
15	FILE WPIDS
15	FILE WPINDEX

L1 QUE LIPASE(10W) UREASE

FILE 'USPATFULL, BIOSIS, CAPLUS, WPIDS, BIOTECHDS, IFIPAT, CABA, EMBASE, FSTA, LIFESCI, TOXLINE, MEDLINE, SCISEARCH, ANABSTR, AQUASCI, BIOBUSINESS, TOXLIT, CEABA, DRUGU' ENTERED AT 17:34:49 ON 04 AUG 1998

L2 201 S LIPASE(10W)UREASE
L3 141 DUP REM L2 (60 DUPLICATES REMOVED)

INDEX 'AGRICOLA, AIDSLINE, ANABSTR, AQUASCI, BIOBUSINESS, BIOSIS, BIOTECHABS, BIOTECHDS, CABA, CANCERLIT, CAPLUS, CEABA, CEN, CIN, CJACS, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGLAUNCH, DRUGNL, DRUGU, EMBAL, EMBASE, FSTA, GENBANK, ...'
ENTERED AT 17:39:44 ON 04 AUG 1998

SEA FAT ABSORPTION AND LIPASE

16	FILE AGRICOLA
3	FILE BIOBUSINESS
90	FILE BIOSIS
0*	FILE BIOTECHABS
1	FILE BIOTECHDS
54	FILE CABA
2	FILE CANCERLIT
74	FILE CAPLUS
8	FILE CJACS
2	FILE DDFB

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                0*  FILE DDFU
                7   FILE DGENE
                2   FILE DISSABS
                2   FILE DRUGB
                1   FILE DRUGNL
               24   FILE DRUGU
                3   FILE EMBAL
              101   FILE EMBASE
                1   FILE FSTA
                2   FILE IFIPAT
                3   FILE LIFESCI
               99   FILE MEDLINE
                1   FILE NTIS
                2   FILE PHAR
                2   FILE PHIN
               63   FILE SCISEARCH
               10   FILE TOXLINE
               16   FILE TOXLIT
               34   FILE USPATFULL
                5   FILE WPIDS
                0*  FILE WPINDEX
L4               QUE FAT ABSORPTION AND LIPASE
                 ---------

     FILE 'EMBASE, MEDLINE, BIOSIS, CAPLUS, SCISEARCH, CABA, USPATFULL,
     DRUGU, AGRICOLA, TOXLIT, TOXLINE, CJACS, DGENE, WPIDS, BIOBUSINESS,
     EMBAL, LIFESCI, CANCERLIT, DISSABS, DRUGB, IFIPAT, PHAR, PHIN,
     BIOTECHDS, DRUGNL, FSTA, NTIS' ENTERED AT 17:49:37 ON 04 AUG 1998
L5             102 S FAT ABSORPTION(25W)LIPASE
L6              55 DUP REM L5 (47 DUPLICATES REMOVED)
L7               0 S L5 AND ANTIBOD?(25W)LIPASE

     INDEX 'AGRICOLA, AIDSLINE, ANABSTR, AQUASCI, BIOBUSINESS, BIOSIS,
     BIOTECHABS, BIOTECHDS, CABA, CANCERLIT, CAPLUS, CEABA, CEN, CIN,
     CJACS, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
     DRUGLAUNCH, DRUGNL, DRUGU, EMBAL, EMBASE, FSTA, GENBANK, ...'
     ENTERED AT 18:08:35 ON 04 AUG 1998
                SEA TETRAHYDROLIPSTATIN AND OBESITY
               ---------
               4    FILE BIOBUSINESS
               4    FILE BIOSIS
               0*   FILE BIOTECHABS
                SEA FAT REDUCTION AND PASSIVE IMMUNITY
               ---------
               0*   FILE BIOTECHABS
               1    FILE CAPLUS
               0*   FILE DDFB
               0*   FILE DDFU

     FILE 'CAPLUS' ENTERED AT 18:20:19 ON 04 AUG 1998
L8               1 S FAT REDUCTION AND PASSIVE IMMUNITY

     FILE 'BIOBUSINESS' ENTERED AT 18:21:48 ON 04 AUG 1998
L9               4 S TETRAHYDROLIPSTATIN AND OBESITY

     INDEX 'AGRICOLA, AIDSLINE, ANABSTR, AQUASCI, BIOBUSINESS, BIOSIS,
     BIOTECHABS, BIOTECHDS, CABA, CANCERLIT, CAPLUS, CEABA, CEN, CIN,
     CJACS, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
     DRUGLAUNCH, DRUGNL, DRUGU, EMBAL, EMBASE, FSTA, GENBANK, ...'
     ENTERED AT 18:23:50 ON 04 AUG 1998
                SEA LIPASE(10W)ANTIBOD?
               ---------
               4    FILE AGRICOLA
               7    FILE ANABSTR
               2    FILE BIOBUSINESS
             142    FILE BIOSIS
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                0*  FILE BIOTECHABS
               31   FILE BIOTECHDS
               18   FILE CABA
                6   FILE CANCERLIT
              135   FILE CAPLUS
                3   FILE CEABA
                2   FILE CJACS
                1   FILE CONFSCI
                1   FILE CROPB
                0*  FILE DDFB
                0*  FILE DDFU
                1   FILE DGENE
                5   FILE DISSABS
                1   FILE DRUGB
                1   FILE DRUGNL
                1   FILE DRUGU
               84   FILE EMBASE
                1   FILE FSTA
                3   FILE IFIPAT
                9   FILE JICST-EPLUS
                1   FILE KOSMET
               22   FILE LIFESCI
               92   FILE MEDLINE
                1   FILE PROMT
               48   FILE SCISEARCH
                4   FILE TOXLINE
               14   FILE TOXLIT
               38   FILE USPATFULL
               29   FILE WPIDS
                0*  FILE WPINDEX
L10              QUE LIPASE(10W) ANTIBOD?
                 ---------

     FILE 'USPATFULL' ENTERED AT 18:36:12 ON 04 AUG 1998
L11           63 S LIPASE(15W)INHIBIT? AND ANTIBOD?
L12            5 S L11 AND LIPASE(25W)ANTIBOD?
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WER 8 OF 24 AGRICOLA
AN 97:80298 AGRICOLA
DN IND20601809
TI Structure-function relationship of lipoprotein lipase-mediated enhancement of very low density lipoprotein binding and catabolism by the low density lipoprotein receptor. Functional importance of a properly folded surface loop covering the catalytic center.
AU Salinelli, S.; Lo, J.Y.; Mims, M.P.; Zsigmond, E.; Smith, L.C.; Chan, L.
CS Baylor College of Medicine, Houston, TX.
SO The Journal of biological chemistry, Sept 6, 1996. Vol. 271, No. 36. p. 21906-21913
Publisher: Bethesda, Md. : American Society for Biochemistry and Molecular Biology.
CODEN: JBCHA3; ISSN: 0021-9258
NTE Includes references
CY Maryland; United States
DT Article
FS U.S. Imprints not USDA, Experiment or Extension
LA English
AB We examined the structure-function relationship of human lipoprotein lipase (hLPL) in its ability to enhance the binding and catabolism of very low density lipoproteins (VLDL) in COS cells. Untransfected COS cells did not bind to or catabolize normal VLDL. Expression of wild-type hLPL by transient transfection enhanced binding, uptake, and degradation of the VLDL (a property of LPL that we call bridge function). Heparin pretreatment and a monoclonal **antibody** ID7 that blocks LDL receptor-binding domain of apoE both **inhibited** binding, and apoE2/E2 VLDL from a Type III hyperlipidemic subject did not bind. However, LDL did not reduce 125I-VLDL binding to the hLPL-expressing cells, whereas rabbit p-VLDL was an effective competitor. By contrast, LDL reduced uptake and degradation of 125I-VLDL to the same extent as excess unlabeled VLDL or beta-VLDL. These data suggest that binding occurs by direct interaction of VLDL with LPL but the subsequent catabolism of the VLDL is mediated by the LDL receptor. Mutant hLPLs that were catalytically inactive, S132A, S132D, as well as the partially active mutant, S251T, and S172G, gave normal enhancement of VLDL binding and catabolism, whereas the partially active mutant S172D had markedly impaired capacity for the process; thus, there is no correlation between bridge function and lipolytic activity. A naturally occurring genetic variant hLPL, S447 replaced by Ter, has normal bridge function. The catalytic center of LPL is covered by a 21-amino acid loop that must be repositioned before a lipid substrate can gain access to the active site for catalysis. We studied three hLPL loop mutants (LPL-cH, an enzymatically active mutant with the loop replaced by a hepatic lipase loop; LPL-cP, an enzymatically inactive mutant with the loop replaced by a **pancreatic lipase** loop; and C216S/C239S, an enzymatically inactive mutant with the pair of Cys residues delimiting the loop substituted by Ser residues) and a control double Cys mutant, C418S/C438S. Two of the loop mutants (LPL-cH and LPL-cP) and the control double Cys mutant C418S/ C438S gave normal enhancement of VLDL binding and catabolism, whereas the third loop mutant, C216S/ C239S, was completely inactive. We conclude that although catalytic activity and the actual primary sequence of the loop of LPL are relatively unimportant (wild-type LPL loop and **pancreatic lipase** loops have little sequence similarity), the intact folding of the loop, flanked by disulfide bonds, must be maintained for LPL to express its bridge function.

CC T200 Physiology of Human Nutrition
CT binding; catabolism; catalytic activity; cell lines; fibroblasts; lipoprotein lipase; low density lipoprotein; man; molecular conformation; mutants; protein degradation; receptors; stimulation; targeted mutagenesis; uptake; very low density lipoprotein
ST cos cells
RN 9001-62-1 (HEPATIC LIPASE)
9001-62-1 (LIPASE)
9004-02-8 (LIPOPROTEIN LIPASE)
9005-49-6 (HEPARIN)

L

L6 ANSWER 17 OF 55 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.DUPLICATE 9

AN 91247714 EMBASE

TI The lipase inhibitor tetrahydrolipstatin binds covalently to the putative active site serine of pancreatic lipase.

AU Hadvary P.; Sidler W.; Meister W.; Vetter W.; Wolfer H.

CS F. Hoffmann-La Roche Ltd., PF/CVD, 68/309, Grenzacherstrasse 124, CH-4002 Basel, Switzerland

SO J. BIOL. CHEM., (1991) 266/4 (2021-2027).
ISSN: 0021-9258 CODEN: JBCHA3

CY United States

DT Journal

FS 029 Clinical Biochemistry
037 Drug Literature Index

LA English

AB Tetrahydrolipstatin (THL) is a selective inhibitor of **fat absorption**. In animal models, it has anti-obesity and anti-hypercholesterolemic activity and is presently in clinical trials for these indications. THL binds covalently to pancreatic **lipase**. Complete inhibition of lipolytic activity is obtained concomitant with the incorporation of 1 mol of THL/mol of enzyme. Pancreatic lipase is the best studied lipase, but published results concerning its catalytic mechanism are still controversial. In order to learn more about the inhibitory mechanism of THL, a selective lipase inhibitor interacting at or near the catalytic site, and therefore, to obtain more information on the catalytic mechanism of lipase, we have determined the amino acid residue to which THL is bound. After proteolytic degradation of porcine pancreatic lipase inhibited with radioactively labeled THL, the labeled peptides were isolated and analyzed by quantitative amino acid analysis, N-terminal sequencing, and by mass spectrometry with fast atom bombardment ionization. The data clearly show that THL is bound as an ester to the serine 152 of the lipase.

AB Tetrahydrolipstatin (THL) is a selective inhibitor of **fat absorption**. In animal models, it has anti-obesity and anti-hypercholesterolemic activity and is presently in clinical trials for these indications. THL binds covalently to pancreatic **lipase**. Complete inhibition of lipolytic activity is obtained concomitant with the incorporation of 1 mol of THL/mol of enzyme. Pancreatic lipase. . .

9 ANSWER 78 OF 89 BIOSIS COPYRIGHT 1998 BIOSIS
AN 80:190560 BIOSIS
DN BA69:65556
TI METABOLIC FUNCTION OF HEPARIN RELEASABLE LIVER LIPASE.
AU JANSEN H; VAN TOL A; HULSMANN W C
CS DEP. BIOCHEM. I., MED. FAC., ERASMUS UNIV. ROTTERDAM, P.O. BOX 1738, 3000 DR ROTTERDAM, NETH.
SO BIOCHEM BIOPHYS RES COMMUN 92 (1). 1980. 53-59. CODEN: BBRCA9 ISSN: 0006-291X
LA English
AB IV administration of specific [rabbit] antibody against heparin-releasable [rat] liver **lipase** (liver **lipase**) induced a 75% inhibition of the enzyme activity in situ. Administration of the **antibody** resulted in an increase of high density lipoprotein (density range 1.050-1.13 g/ml; HDL2) phospholipid levels (20% after 1 h; 54% after 4 h). Short-term (1 h) **treatment** with antibody had no significant effect on any of the other lipoprotein components. After long-term (4 h) **treatment** the free cholesterol level of HDL2 and all components in the very low density lipoprotein (VLDL) + intermediate density lipoprotein (IDL) fraction were elevated (1.5-2.0-fold). In the low density lipoprotein (LDL) fraction only the phospholipid level was affected (increased by 72%). All lipid components in the HDL3 fraction were decreased by the antibody **treatment**, but this decrease was only statistically significant for the cholesterolesters. The removal rate of iodine-labeled high density lipoprotein (HDL) and LDL from serum was not affected by the antibody **treatment**. Liver lipase may promote phospholipid removal in vivo. A lowering of liver lipase in situ apparently has profound consequences for serum lipoprotein metabolism.

L

L8 ANSWER 1 OF 1 CAPLUS COPYRIGHT 1998 ACS
AN 1996:423154 CAPLUS
DN 125:83777
TI **Fat reduction** through the use of **passive immunity**
AU Brodie, A.; Hu, C. Y.
CS Department Animal Sciences, Oregon State University, Corvallis, 97331-6702, USA
SO Biol. Fat Meat Anim. (1995), 70-77. Editor(s): Smith, Stephen B.; Smith, D. R. Publisher: American Society of Animal Science, Champaign, Ill.
CODEN: 63AQA9
DT Conference; General Review
LA English
CC 15-0 (Immunochemistry)
AB A review with 34 refs. on use of **passive immunity** in relation to fat redn. in domestic meat-producing animals. Topics discussed include **passive immunity** against plasma membrane protein; **passive immunity** against growth hormone or somatostatin;.
ST review fat redn meat animal immunity
IT Adipose tissue
(use of **passive immunity** to reduce fat in domestic animals)
IT Animal
(domestic, use of **passive immunity** to reduce fat in domestic animals)
IT Immunity
(passive, use of **passive immunity** to reduce fat in domestic animals)

9 ANSWER 45 OF 89 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 13
AN 89:423439 BIOSIS
DN BA88:81697
TI BIOSYNTHESIS OF LIPOPROTEIN **LIPASE** IN CULTURED MOUSE ADIPOCYTES I. CHARACTERIZATION OF A SPECIFIC **ANTIBODY** IN RELATIONSHIPS BETWEEN THE INTRACELLULAR AND SECRETED POOLS OF THE ENZYME.
AU VANNIER C; DESLEX S; PRADINES-FIGUERES A; AILHAUD G
CS EMBL, POSTFACH 110.2209, MEYERHOFSTRASSE 1, 6900 HEILDELBERG, FRG.
SO J BIOL CHEM 264 (22). 1989. 13199-13205. CODEN: JBCHA3 ISSN: 0021-9258
LA English
AB Polyclonal antibodies have been raised in rabbits against homogenous lipoprotein **lipase** (LPL) purified from the media of adipose 3T3-F442A cells. The **antibody** is able to inhibit the apolipoprotein C-II-dependent activity of LPL, to immunoprecipitate LPL under nondenaturating conditions from media and cellular extracts. A dot-blot immunoassay of secreted LPL is also described (range 0.1-0.7 melliunits). The secretion potential .mu., taken as the ratio of total releasable activity or antigen to initial cellular activity or antigen, was determined. This was shown in cells **treated** with heparin and cycloheximide to be equal to 1 for LPL antigen but significantly greater than 1 for LPL activity assayed under standard conditions. No LPL was actually degraded within the cells. A dramatic enhancement of the intracellular activity was induced by a mere dilution of detergent-**treated** cell lysates with no change in LPL antigen. The total intracellular activity reached a plateau at a value which now became identical to that obtained in the medium of cells exposed to heparin and cycloheximide. The existence of an inhibitor of LPL activity has been excluded as well as that of an increase in the catalytic activity of LPL during its secretion, before or after exposure to heparin. Our results indicate a systematic underestimation of LPL intracellular activity and suggest that LPL is present within intracellular cisternae in a cryptic state. This potetial activity can be fully unmasked in vitro. In agreement with other data (Vannier, C., and Ailhaud, G., (1989) J. Biol. Chem. 264, 13206-13216), our results appear to exclude the existence of a reservoir of catalytically inactive LPL molecules within adipose cells.

L

L12 ANSWER 4 OF 5 USPATFULL
AN 90:59329 USPATFULL
TI Dietary compositions and methods using bile salt-activated lipase
IN Tang, Jordan J. N., Oklahoma City, OK, United States
Wang, Chi-Sun, Oklahoma City, OK, United States
PA Oklahoma Medical Research Foundation, Oklahoma City, OK, United States (U.S. corporation)
PI US 4944944 900731
AI US 87-122410 871119 (7)
DT Utility
EXNAM Primary Examiner: Stone, Jacqueline
LREP Kilpatrick & Cody
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 586
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Dietary compositions, especially cow's milk-based infant formulas, are fortified with bile salt-activated lipase. Methods are provided for feeding newborn and premature infants which include administration of bile salt-activated lipase to increase fat digestion and therefore growth rate. Similarly, a method is provided to treat subjects for inadequate pancreatic enzyme production by administration of bile salt-activated lipase in conjunction with ingestion of fats.

=> d l12 4 kwic

L12 ANSWER 4 OF 5 USPATFULL
SUMM As naturally occurring BAL has been isolated, anti-BAL **antibodies** may be produced and used to find the BAL clones in the expression libraries. Alternately, a partial structure of the. . .
DETD . . . these enzymes had not been demonstrated. Accordingly, we next examined the cross-reactivity of human BAL and cat BAL by performing **antibody** inhibition studies.
DETD **Antibodies** against human bile salt-activated **lipase** were prepared from a rabbit. The **antibodies** in the antiserum from the rabbit was collected and purified using affinity chromatography. Specifically, we used an affinity column loaded with covalently linked purified human BAL and Sepharose 4B. 3 M NaSCN was used to elute the retained **antibodies**. The monospecific **antibodies** then were used in a **lipase** assay procedure to test reactivity of the **antibodies** with human milk bile salt-activated lipase and with cat milk bile salt-activated lipase. The results are shown in Table II.
DETD TABLE II

EFFECT OF HUMAN MILK BAL **ANTIBODIES** ON BAL ACTIVITY IN HUMAN MILK, CAT MILK AND **ANTIBODY**-FREE SERUM

Control (Non-BAL immunized rabbit serum gamma globulin) **Antibody** Aliquots (ml)	% Activity Remaining	CPM*	BAL Activity **

0.000	100.0	2001	290.29
0.025	100.9	2019	292.90
0.050	99.6	1994	289.29
0.100	97.8	1957	283.91
0.150	98.0	1961	284.45
0.200	96.9	1938	281.15

Cat Milk
Antibody

Aliquots (ml)	% Activity Remaining	CPM*	BAL Activity**
0.000	100.0	2001	290.29
0.025	87.3	1747	253.44
0.050	74.5	1491	216.30
0.100	45.4	908	131.73
0.150	32.9	659	95.60
0.200	23.7	475	68.91

Human Milk
Antibody

Aliquots (ml)	% Activity Remaining	CPM*	BAL Activity**
0.000	100.0	993	144.06
0.025	41.1	408	59.19
0.050	15.0	149	21.62
0.100	5.4	54	7.83
0.150	5.1	51. . .	

DETD As Table II shows, **antibodies** against bile salt-activated **lipase** from human milk **inhibited** enzyme activity in both cat milk and human milk. However, the human enzyme **antibodies** were only about 70% as reactive with the cat enzyme as with the human enzyme. From this we concluded that. . .